ON THE RELATION OF

Diseases of the Spinal Cord

TO THE DISTRIBUTION AND LESIONS
OF THE

Spinal Blood Vessels.

BY

R. T. WILLIAMSON, M.D. (Lond.), M.R.C.P.,

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PREFACE.

THE following pages consist of a reprint (with a few additions) of papers which have been already published in the *Medical Chronicle* for December, 1894, and January and February, 1895.

No attempt has been made to review the subject exhaustively, and the consideration of several rare diseases of the spinal cord has been omitted, since the records of the pathological changes are as yet few and incomplete.

To the kindness of Dr. Graham Steell, the late Dr. Ross, Dr. Pullon, of Burnley, and Dr. Wilson, of Cheadle, I am indebted for the opportunity of making the pathological examination in some of the cases described.

R. T. W.

ON THE RELATION OF SPINAL DISEASES TO THE DISTRIBUTION AND LESIONS OF THE BLOOD VESSELS OF THE SPINAL CORD.

A VERY large proportion of cases of cerebral diseases, excluding cases of insanity, are due to vascular lesions. According to Dr. Hughling Jackson* nearly all the diseases of the brain, of which we *know* the morbid anatomy, are not nervous diseases in the strict sense. They are "damages of the nervous organs"; the morbid changes, however, do not begin in the nervous elements, but in other structures—connective tissue, blood vessels, etc. It appears, therefore, somewhat surprising that so little importance, comparatively speaking, has been attached to vascular lesions as a cause of diseases of the spinal cord.

There can be no doubt that cerebral affections are much more frequently the result of vascular lesions than diseases of the spinal cord, and this is due chiefly to the differences in the vascular supply and distribution in these two parts of the nervous systems. Nevertheless, it is very probable that vascular lesions play a much more important part in the pathology of spinal diseases than has been attributed to them in the past; and the object of the following pages is to consider the relation of a number of diseases of the spinal cord (1) to the distribution and (2) to the lesions of the spinal vessels.

It will be necessary, in the first place, to refer to the distribution of the spinal vessels, and to mention some peculiarities of the blood supply of the cord.

I.

THE BLOOD VESSELS OF THE SPINAL CORD.†

The spinal cord is supplied by narrow, long and tortuous arteries, and hence the vessels are not subject to high pressure, which is such an important cause of degeneration and rupture in the arteries of the brain.

The spinal cord derives its blood supply through small branches coming from the vertebral, intercostal, lumbar, and sacral arteries. A

* *British Medical Journal*, July 21st, 1888, page 115.

† ROSS.—*Brain*, Vol. III., April, 1880, p. 80. OBERSTEINER.—"Anleitung beim Studium des Baues der Nervösen Centralorgane," p. 197. Liepzig, 1888. KADYI.—*Jahresbericht über die Fortschritte der Anatomie und Physiologie*, 1886, Bd. XV. (Original paper.) "Anatom. Anzeiger," No. 12, p. 304. ADAMKIEWICZ.—*Transactions International Medical Congress, London*, 1881, Vol. I., p. 155. MARIE.—"Leçons sur les Maladies de la Moelle." Paris, 1892. GOWERS.—"Diseases of the Nervous System," Vol. I., p. 187. London, 1892. SCHÄFER.—"Quain's Anatomy."

slender branch arises from each vertebral before these arteries unite to form the basilar. These two slender arterial branches pass to the ventral side of the medulla and unite at the upper end of the cervical region of the cord to form a single artery—the *anterior spinal*, which runs downward in the region of the anterior fissure to the conus medullaris. (Sometimes the anterior spinal artery arises from one vertebral only.) The branches from the vertebral, intercostal, lumbar, and sacral arteries pass into the spinal canal through the intervertebral foramina, and run along the anterior and posterior nerve roots. The branches accompanying the anterior nerve roots, for the most part, join the anterior spinal artery at the anterior median fissure. The anterior spinal artery furnishes branches to the anterior roots, and some anastomotic branches, passing to the posterior spinal system of arteries.

From each vertebral artery a small branch winds round the side of the medulla to the posterior aspect of the cord. These two branches, the *posterior spinal arteries*, do not unite, but pass down to the cauda equina, one on each side of the cord, just in front (*i.e.*, on the outer side) of the posterior nerve roots. These arteries receive numerous communicating branches from the intercostal and lumbar arteries. Just behind (to the inner side) of the line of the posterior nerve roots, is another small longitudinal anastomotic chain, formed by branches of the posterior spinal.

Branches from the anterior and posterior spinal arteries ramify in the pia mater of the cord and form transverse anastomoses. From the main arteries and their ramifications branches pass into the cord to supply the grey and white substance.

At the region of the conus medullaris the arteries are remarkably tortuous.

The blood vessels passing into the cord substance (intra-medullary branches) may be divided into two groups : those from the anterior and those from the posterior system of spinal arteries. From the anterior spinal artery frequent branches pass horizontally (and therefore at right angles to the artery) into the anterior median fissure. These arteries are known as the arteriæ sulci or the *anterior median arteries.* According to Adamkiewicz, in an adult man they are about 250 to 300 in number. At the bottom of the anterior median fissure each artery turns either to the right or to the left, enters the anterior commissure, and passes to the grey matter. (In the older descriptions, the anterior median artery is generally stated to divide, at the bottom of the anterior fissure, into two branches. According to Kadyi, bifurcation is very rare.) When the anterior median artery enters the commissure it is known as the *commissural artery.* It passes outwards and backwards, and at the outer part of the grey commissure divides into : (*a*) the *anterior central* artery,

which passes to the anterior horn; and (*b*) the *posterior central*, which passes to the intermediate grey matter, the neck of the posterior horn, and to the posterior vesicular column of Clarke. Each commissural artery gives off, just before dividing, a small branch (*c*), the *anastomotic*, which bifurcates at once. One branch passes upwards to unite with a branch from the next commissural artery above; the other passes downwards and joins a branch from the commissural artery below; a vertical anastomosis is thus formed within the grey matter of the spinal cord. Some branches pass from the anterior median artery to the white substance in the neighbourhood of the anterior horns of grey matter. The distribution of the anterior median artery and its branches is chiefly to the *central part of the grey matter* of the spinal cord. The following arteries are distributed chiefly to the *peripheral parts* of the cord, the white matter. Short branches pass from the anterior spinal artery to the anterior part of the white substance; longer branches from this artery pass through the anterior white matter and end in the grey anterior horn. Small arteries—*anterior root arteries*—accompany the fibres of the anterior roots into the interior of the cord; they supply branches to neighbouring white matter, and terminate in the anterior grey horns. (The above-mentioned branches all proceed from the anterior system of spinal arteries.) Small branches pass from the periphery into the lateral white matter of the cord. Three of the largest of these are named the *anterior*, *middle*, and *posterior lateral* arteries respectively, and their course is indicated in Fig. 1. These three arteries, especially the anterior lateral, are in part furnished by the anastomotic branches between the anterior and posterior system of arteries; but they depend chiefly on the posterior system.

The following arterial branches are also supplied by the posterior system of arteries:—

A branch, the *posterior median*, courses in the posterior median septum, almost up to the grey matter, and gives numerous small branches to the columns of Goll on each side. Another branch, the *intermediate septal artery* (art. interfunicularis), runs in the septum between the columns of Goll and Burdach. A little to the outer side of this is a branch which runs through Burdach's column to the caput of the posterior horn—the *posterior cornual artery*. Entering the cord on the inner (median) side of the posterior root is the *posterior radicular* artery. This is distributed to the caput and the posterior root.

The vessels supplying the cord may, therefore, be divided into two arterial systems:—

(1) The *central arteries*—the *anterior median arteries*—entering at the anterior median fissure, and supplying the grey matter, with the exception of the caput and posterior horn.

(2) The *peripheral arteries—vaso-corona* (Adamkiewicz). These are the small arteries running from the periphery of the cord into the white matter, *i.e.*, towards the centre. The smaller branches end in the white matter, whilst the larger ones extend to the grey matter.

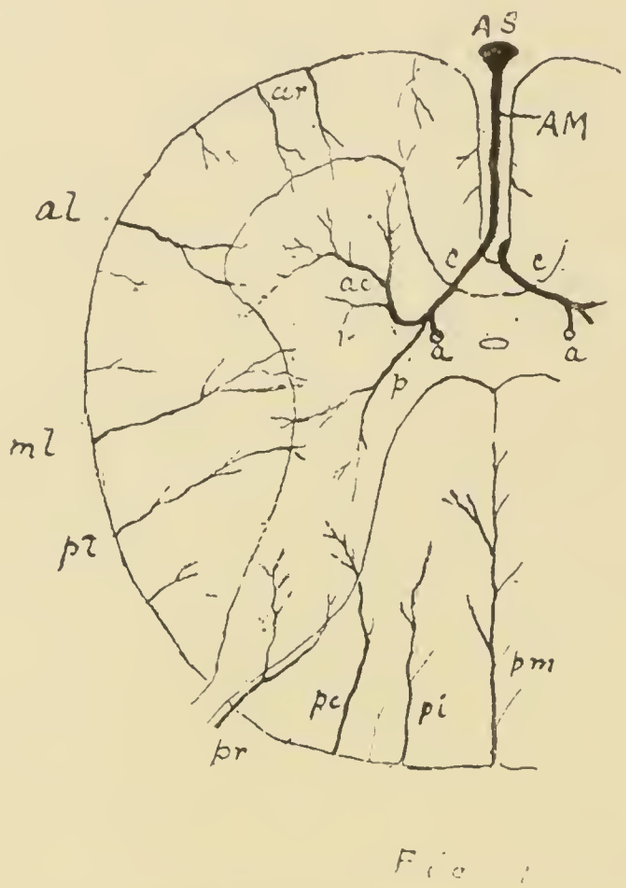

FIG. 1.—Arteries of the Spinal Cord (modified after Obersteiner).

A S=Anterior Spinal.
A M=Anterior Median.
c=Commissural.
a=Anastomotic.
a c=Anterior Central.
p=Posterior Central.
a r=Anterior Root Arteries.
a l=Anterior Lateral.
m l=Median Lateral.
p l=Posterior Lateral.
p r=Posterior Root Artery.
p c=Posterior Cornual.
p i=Intermediate Septal.
p m=Posterior Median.

But there is no sharp division between these two arterial districts of the cord, since the peripheral part of the grey matter and the adjacent white matter receive arteries from both systems. Hence a transverse section of the cord may be mapped out into three districts (see Fig. 2), according to the arterial supply :—

(1) The inner part of the grey matter (with the exception of the posterior horn and the caput), which is supplied exclusively by the anterior median artery (area shaded with parallel lines in Fig. 2).

(2) The superficial part of the white matter, which is supplied by the peripheral arteries (dotted area in Fig. 2).

(3) The peripheral part of the grey matter and the adjacent white matter, which receive arteries from both the peripheral and central systems (area not shaded in Fig. 2).

This district, common to the two arterial systems, forms about one-third of the total area of the transverse section of the cord (Kadyi)

The blood supply of the cord may also be divided into two systems, the anterior and posterior arterial systems; and the transverse area of the cord may be divided into two districts, according to the origin of the supplying arteries from the anterior or posterior arterial system.

In Fig. 3 (modified after Marie), the dotted area represents the part

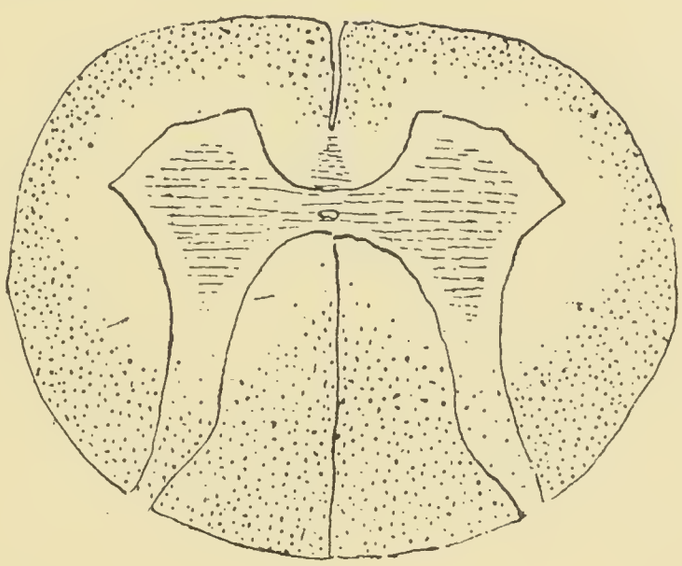

Fig 2

Fig. 2.—Section of the Cord showing division into three arterial districts (diagrammatic). Part supplied only by the Anterior Median and its branches is shaded with parallel lines. Part supplied only by the Peripheral Arteries is shaded with dots. Part supplied by both systems of Arteries is unshaded.

supplied by branches from the *posterior* arterial system; the unshaded area, that supplied by the *anterior* system.

The grey matter is more richly supplied with blood than the white. The white substance has only a relatively scanty zone of vessels, whilst the grey matter possesses a thick network of capillaries. The greater quantity of blood passing into the spinal cord enters the grey matter.

According to Kadyi, the arteries penetrating the spinal cord are true terminal arteries (as defined by Cohnheim) and do not anastomose.

Dr. Moxon* some years ago drew attention to the feeble blood supply of the lowest part of the spinal cord. The anterior and posterior spinal arteries are very slender vessels, they have a long course from the verte-

* Moxon. *Lancet*, Vol. I., 1881, p. 530.

*

brals to the lower end of the eord, and no arteries so small as these run so long a course elsewhere in the body. The reinforeing arteries passing along the nerve roots in the upper part of the cord are short and run almost horizontally. At the lower end of the eord, the supply from above by the anterior and posterior spinal arteries is made with greatest difficulty owing to the distanee from the vertebral. Further, since the eord ends at the level of the second lumbar vertebra, the reinforeing arteries which run along the nerve roots to the lumbar enlargement must aseend for a considerable distance before reaching the eord. The reinforcing arteries which run along the nerve trunks of the eauda equina to the lower part of the cord are of great length ; they are very. narrow ;

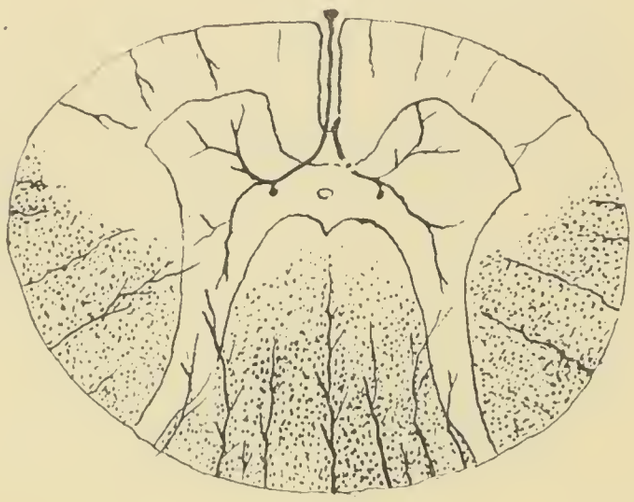

Fig. 3

FIG. 3.—Transverse section of Cord showing distribution of Anterior and Posterior Arteries modified after Marie). Part supplied by Posterior Arterial system shaded with dots. Part supplied by Anterior system unshaded. (The diagram is not quite correct, as the posterior horn ought to have been included in the dotted area as in Fig. 2).

also they are very tortuous. Hence the blood supply from below (as well as that from above) is very feeble, and at a diminished pressure in the lower part of the cord.

At the request of Dr. Moxon, Dr. Carrington injeeted seven subjects in the dissecting room, in order to investigate the blood supply of the lower part of the eord. The subjeets were injected from the femoral artery, and therefore, *à priori*, there would be a greater probability of the lower part of the cord being well injected. But this was not the case. In all the cords there was elear evidence that the anterior spinal artery filled from above downwards, the injeetion eeasing at the lower end of the cord or above that part. It was obvious that reinforcing vessels entered along the nerves. In no case were the reinforeing vessels of the eauda equina injected.

Veins of the Spinal Cord.—The veins anastomose freely, and to the returning blood many paths are open. On the anterior surface is a longitudinal vein following the course of the anterior median fissure, with the anterior spinal artery. Another longitudinal vein follows the course of the posterior median fissure, but is unaccompanied by any considerable artery. The course of both of these veins is tortuous, and the posterior vein is frequently broken up into a kind of venous plexus, with longitudinal meshes extending over the posterior surface of the cord. Behind the line of exit of the anterior nerve roots there is a less perfect lateral anastomotic chain. By means of lateral offsets these vessels communicate freely.

These veins receive the venous blood from the cord and deliver it by communicating veins into large venous plexuses external to the dura mater. Blood passes into these plexuses from the vertebræ and from the skin and adjacent tissues behind the spine. From the plexuses blood is carried away to the cervical and intercostal veins, by branches which run at frequent intervals along the nerve roots. At the upper part these plexuses join the vertebral veins. Dr. Gowers points out, that the veins of the cord cannot be injected from the extra-dural plexuses, apparently because they form so trifling a proportion of the total connections; and the conditions that prevent an injection of the plexus of veins, outside the dura mater, from passing into the veins of the cord, must save the latter from the extreme over distension, to which they would otherwise be liable, when there is a hindrance to the return of blood from the plexus.

The course of the veins *within* the cord to some extent corresponds to that of the arteries. Venæ comitantes follow the anterior arterial chain only; otherwise the arteries and veins course apart from each other (Kadyi). A larger portion of the blood passes into the peripheral veins, especially into the posterior veins, than into those accompanying the anterior median arteries. The *anterior arteries* are more important than the posterior, but the *posterior veins* are more important than the anterior.

There is a point of interest and possibly of considerable pathological importance with regard to the *lymphatics* of the spinal cords. There are no true lymphatic vessels in the cord; but external to the adventitia of the spinal blood vessels, and ensheathing the vessels, are lymphatic spaces bounded by a limiting membrane—perivascular lymphatics. There are also lymphatic clefts in the adventitia.

II.

The above-mentioned facts, concerning the blood supply of the spinal cord, furnish an explanation of many points with respect to the localisation of lesions in various parts of the white and grey matter.

(A) *Poliomyelitis.*—A spinal cord which I examined some time ago, presented a beautiful example of the localisation of a lesion to the part supplied by the central arteries—*i.e.*, the *anterior median arteries* and their branches.

The case* was one of exceedingly sudden onset. The patient, F. K., was a girl, aged 13. On the day on which her symptoms commenced she arose, as usual, about eight o'clock in the morning, feeling quite well. She dressed herself, combed her hair, and then went downstairs and commenced to wind up the window blinds. She wound up half of them, but on going to wind up the blind of the next window she found that she could not extend the arms. Both arms had suddenly become paralysed, and (according to the patient, who was a most intelligent girl) the parts paralysed were those which were found to be paralysed when she first came under observation at the Infirmary. About an hour later, the legs became feeble and by evening were completely paralysed. There was retention of urine, left-sided analgesia and thermo-anæsthesia up to the 7th rib, but tactile sensation was not affected. The deltoid, biceps and supinator longus muscles on each side were not affected, but the triceps, the extensors and flexors of the wrist, the extensors of the fingers, and the small muscles of the hands were paralysed completely on both sides, the flexors of the fingers almost completely. Death occurred at the end of 39 days. By its sudden onset the case simulated spinal hæmorrhage, but most careful examination of the cord failed to reveal the slightest trace of hæmorrhage. In the lower half of the cervical region, and the upper two-fifths of the dorsal region, softening—so-called myelitis—was found, chiefly in the grey matter. Where the lesion was most extensive, in the lower cervical region, it involved chiefly the anterior and intermediate parts of the grey matter on each side. It extended more posteriorly on the left side—the side of analgesia and thermo-anæsthesia—than on the right side. In the middle of the affected part, the lesion was situated in, and corresponded to, the region of distribution of the central arteries of the cord—the anterior median and their branches. The anterior median arteries were

* The case is described in detail in the *Lancet*, January 21st, 1893, p. 142. (During life the patient was under the care of Dr. Steell at the Manchester Royal Infirmary.)

greatly dilated and surrounded by round cells (in the anterior fissure). In the grey matter, at the affected part, the vessels were greatly dilated, their perivascular sheaths were distended with round cells, and the tissue densely infiltrated with leucocytes and compound granular cells.

In Fig. 2, the part supplied only by the anterior median arteries is shaded with parallel lines, the part supplied both by branches of the anterior median and by the peripheral arteries is unshaded. Now, in the case to which I am referring, the myelitis corresponded at the seat of greatest intensity (*i.e.*, in the lower cervical region) to the shaded area of Fig. 2, in many places it invaded the unshaded area, and only at a few points extended slightly beyond this area. At the point of greatest intensity the lesion was, broadly speaking, localised to the region supplied by the anterior median arteries and their branches.

Fig. 4 represents the appearance (diagrammatically) of a transverse section of the cord in the lower cervical region, stained according to Weigert's method. The black dots represent the healthy nerve fibres of the white matter, the fine black lines the healthy nerve fibres of the grey matter. The clear space represents the area of myelitis (or softening). A comparison of these diagrams will show, how closely the clear area of myelitis in Fig. 4 corresponds with the area shaded by parallel lines and part of the clear area of Fig. 2—*i.e.*, the region supplied by the anterior median arteries. At the upper dorsal region the changes were chiefly around the branches of the anterior median arteries at the commissure and in the anterior horns. (The cause of the softening in this case will be discussed later.)

(B) On the other hand, it is interesting to note how, in *hereditary ataxia* (Friedreich's disease) the area of degeneration, at those parts of the cord where the lesion is most intense, corresponds roughly to the part of the transverse section supplied by the *peripheral arteries of the cord* (the vaso-corona).

Fig. 5 represents a transverse section of the cord in the lower cervical region in a case of Friedreich's disease. The dotted area is that of degeneration. A comparison of Figs. 2 and 5 will show that this area of degeneration in Friedreich's disease corresponds roughly with the dotted area in Fig. 2—the area of distribution of the peripheral arteries.

(C) In Fig. 3 the dotted area represents the part supplied by the posterior arterial system of the cord. Marie has pointed out, that in

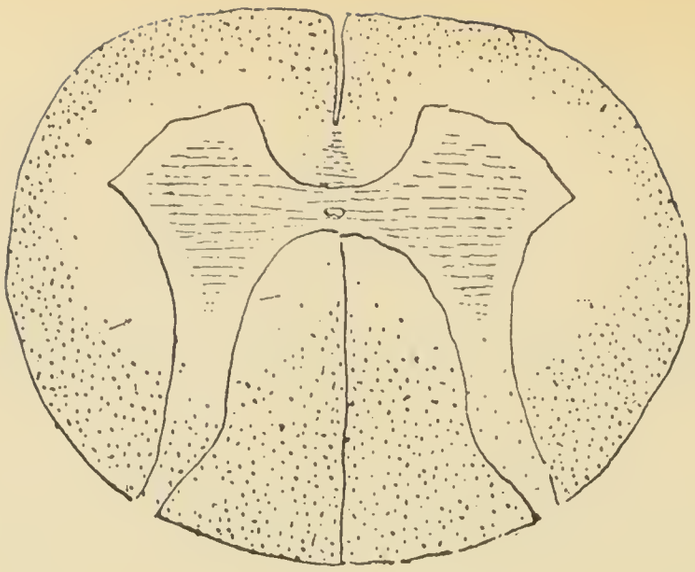

Fig 2

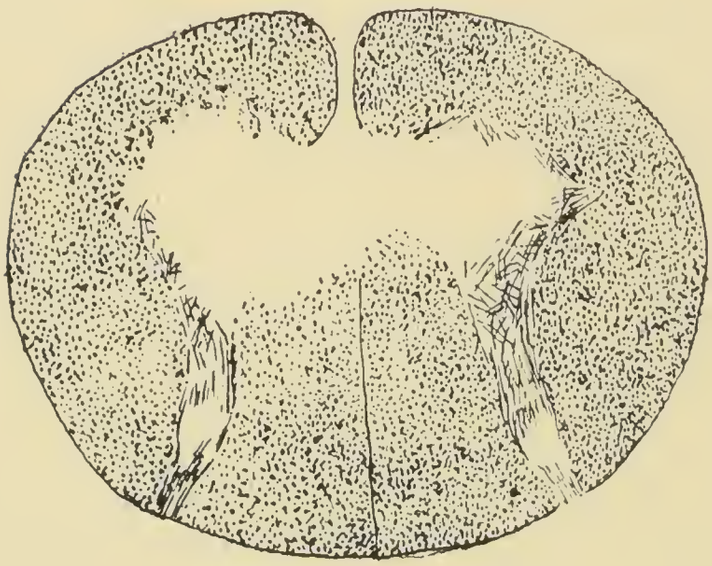

Fig. 4.

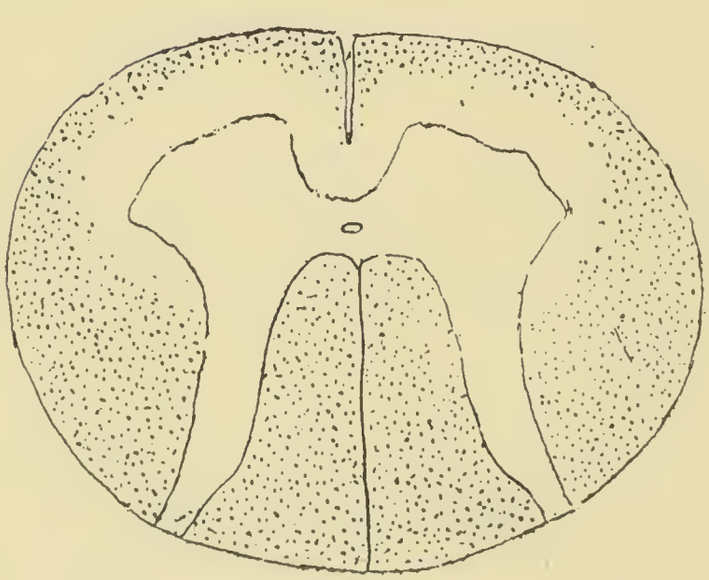

Fig. 5

FIG. 2.—Transverse section of Normal Cord. Area shaded with parallel lines=part supplied by Anterior Median Arteries and branches. Area unshaded=part supplied both by branches of Anterior Median and by Peripheral Arteries of the Cord. Area shaded by dots=part supplied only by Peripheral Arteries.

FIG. 4.—Section of Cord in the case F. K.–poliomyelitis. Weigert's stain (diagrammatic) Black dots=healthy nerve fibres; black lines=healthy fibres in grey matter. Unshaded blank part=area of myelitis.

FIG. 5—Cervical Cord in Hereditary Ataxia (modified after Friedreich). Area of degeneration is shaded with black dots.

certain cases of *combined postero-lateral sclerosis*, the sclerotic area corresponds pretty closely with that supplied by the *posterior arterial system* of the cord.

In Fig. 6 the shaded part represents the area of sclerosis, at the region where the sclerosis was most marked, in a case of postero-lateral

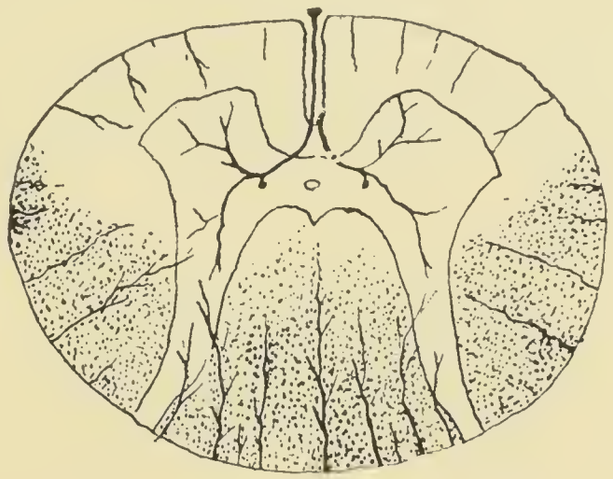

Fig. 3

FIG. 3—Section showing distribution of Anterior and Posterior Arterial system of the Cord. Part shaded by dots = area supplied by Posterior Arteries of the Cord.

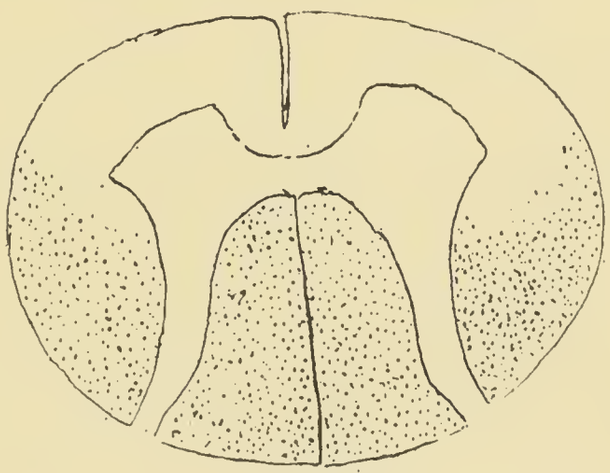

Fig. 6

FIG. 6.—Section of the Cord in a case of combined Postero-lateral Sclerosis. Part shaded with dots = area of degeneration (modified after Ballet and Minor).

sclerosis, reported by Ballet and Minor,* and a comparison of Figs. 3 and 6 will show, how closely the shaded area of degeneration in Fig. 6 corresponds to the dotted area of the distribution of the vessels of the posterior system of the cord in Fig. 3.

* BALLET and MINOR.—"Archives de Neurologie," 1884, Tome VII., p. 4

From the changes found in the vessels, Ballet and Minor believe the disease to have been due to a perivascular sclerosis. Marie regards vascular changes in the cord as the most frequent cause of postero-lateral sclerosis.

Ehrlich and Brieger* have repeated Stenson's well-known experiment [by which paralysis was produced through temporarily suspending the circulation in the lower part of the cord by compressing the abdominal aorta], and have studied the changes thereby produced in the spinal cord. They have found that the grey substance and the antero-lateral columns present considerable degeneration, whilst the posterior columns are almost intact, with the exception

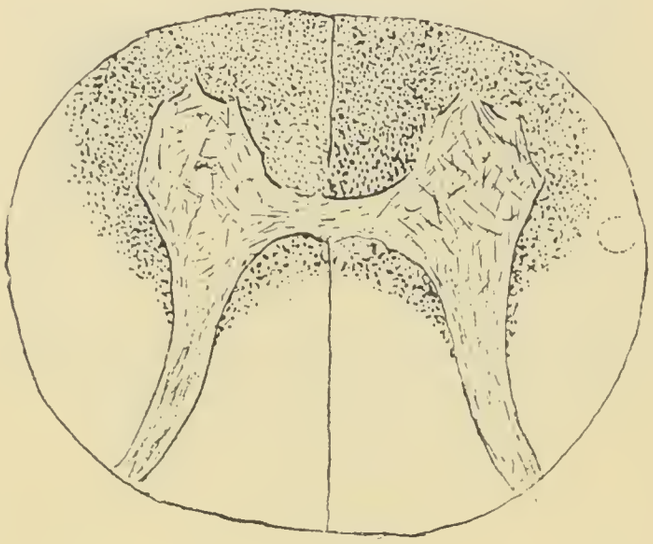

FIG. 6A.—Section of spinal cord, Weigert's stain (diagrammatic). The black dots and lines= healthy nerve fibres; the blank area=softening and degeneration.

of their anterior borders close to the posterior commissure. Singer and Münzer have shown, that after this experiment a marginal zone is preserved in the lateral columns corresponding to the direct cerebellar tracts and the ascending antero-lateral tracts of Gowers. Marie† points out that the conclusion to be drawn from these experiments is, that the columns of the cord may be divided into two groups in the lower part. In one group—in the antero-lateral columns and the grey substance—the nutrition depends directly on the circulation in the abdominal aorta. In the other group—the posterior columns, the direct cerebellar tracts, and the columns of Gowers—the nutrition of the nerve elements independent of the circulation in the abdominal aorta.

* *Zeitschrift f. klin Med.* Supplement to Bd. VII., 1884.

† MARIE.—"Les Maladies de la Moelle." Paris, 1892, p. 413.

A spinal cord which I had recently the opportunity of examining showed beautifully the localisation of the lesion to the part of the transverse section supplied by the posterior spinal arteries. The case was one of myelitis (or, perhaps, it would be more correct to describe it as softening) of the lower dorsal region of the cord. At this part the lesion implicated the posterior columns (with the exception of a narrow zone close to the posterior commissure), the lateral pyramidal tracts, the direct cerebellar tracts, and the periphery of the cord for a short distance in front, whilst the white matter of the anterior part of the cord and the grey matter were not invaded by the softening. Fig. 6A represents, diagrammatically, the appearance of a section, stained according to Weigert's method, the black dots and lines representing the normal nerve fibres. The blank part is the area of softening and degeneration, in which the nerve fibres have been destroyed and the tissues infiltrated with round cells (leucocytes and compound granular cells).

Now a comparison of Fig. 6A with Fig. 3 will show how closely the area of degeneration and softening (blank area) corresponds to the parts supplied by the posterior spinal arteries (area shaded with dots in Fig. 3). The parts supplied by the anterior spinal system of arteries were not invaded by softening.

At no part was there a complete transverse myelitis. Above and below the part where the lesion was most marked, there was the usual ascending and descending secondary degeneration.

Now the clinical history of this case is interesting. The patient was a man, aged 50, who was admitted under the care of Dr. Steell at the Manchester Royal Infirmary, suffering from paraplegia. He had been quite well until three weeks previously, when he suddenly lost the use of his left leg, just before going to bed in the evening. He had just passed urine, and was pulling off his stockings, when he found that he was unable to raise the left leg. The right leg became paralysed soon afterwards. There was no pain in the limbs or back at the onset, but very soon the left leg became painful. Retention of urine soon occurred, and this, with complete paraplegia, continued until his death, five months later. On admission, the knee-jerks were present, but there was no ankle clonus. There was anæsthesia to tactile and painful impressions on the right leg and the lower part of the abdomen, up to a point midway between the pubes and umbilicus; but on the left leg tactile and painful sensations were normal. (Observations made towards the end of his illness were unreliable, owing to the patient's mental condition.)

The localisation of the lesion to the part supplied by the posterior arterial system (whilst the regions supplied by the anterior spinal arteries, *i.e.*, the most vascular parts, were free), together with the very sudden onset, is suggestive of a vascular origin.

**

(D) *Acute anterior poliomyelitis of the infant. Infantile paralysis.*—In many cases of this disease, if the spinal cord be obtained for examination at a very early stage, the anterior horn of grey matter is found to be infiltrated with round cells, the vessels greatly distended, and the perivascular lymph sheaths crowded with round cells, changes which indicate an interstitial inflammation. In some cases hæmorrhagic softening has been found in the anterior horns.

Marie,* Goldscheider, Dauber, Siemerling, and Redlich look upon the disease as one of arterial origin, and have shown that the changes are localised to the part supplied by the anterior spinal artery.

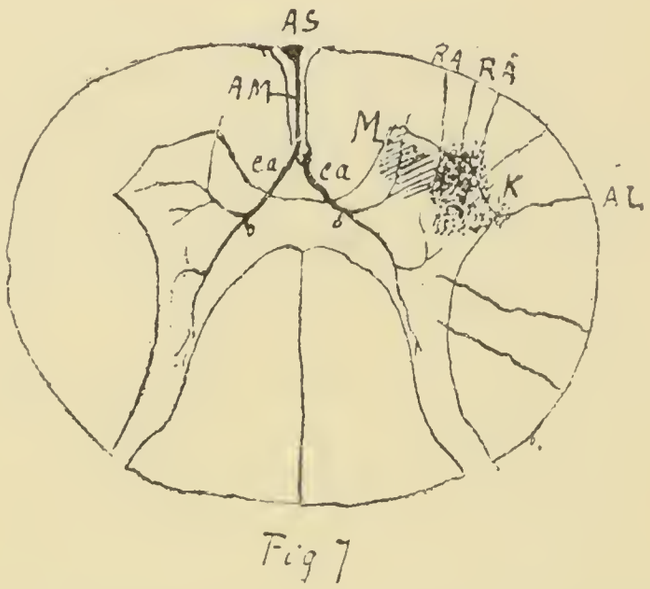

FIG. 7.—Section of the Cord showing two regions at which the changes may be most marked in infantile paralysis. M (area shaded with parallel lines)—part supplied from the Anterior Median (A M) by means of a branch from the Commissural Artery (*c a*). K (area shaded by dots) —part of grey matter in which the Anterior Radicular Arteries terminate (R A). Modified after Marie.

Marie points out that changes are found (*a*) in many cases in the region of distribution of the anterior median artery. Generally they are found around a branch of the commissural artery going to the anterior horn. (*b*) In other cases they are found around the anterior radicular arteries as they enter the anterior horns of grey matter. (In Fig. 7 these two regions are mapped out.)

The changes are found, as a rule, in one anterior horn only, because, as Marie points out, the anterior median artery does not divide (except very rarely), but passes only to one anterior horn. Hence a lesion in the

MARIE. *Loc. cit.*, p. 455. GOLDSCHEIDER. "Ueber poliomyelitis."—*Zeitschrift für klin sche Medicin*, Bd. XXII., Heft 5 und 6. DAUBER. "Zur Lehre von der Poliomyelitis anterior acuta."—*Deutsche Zeitschrift für Nervenheilkunde*, Bd. IV., p. 200. SIEMERLING. "Zur pathologischen Anatomie der Spinalen Kinderlähmung."—*Archiv für Psychiatrie*, Bd. XXVI., Heft 1.

distribution of *one* anterior median artery would be followed by change n *one* anterior horn, and this explains why acute anterior poliomyelitis of the infant generally causes unilateral paralysis.

Sometimes the pathological changes extend to the white matter around the anterior horn. This may be explained by the fact that the branches of the anterior median are not all limited to the anterior grey matter. Terminal branches pass to the white matter just around the anterior horn.

The frequent occurrence of infantile paralysis after or during the progress of an infectious malady, as well as the occasional apparent epidemics of the disease, show that it is often closely related to infectious ailments. The exceedingly sudden onset, in some cases presenting the usual symptoms of infantile paralysis, has been suggestive either of hæmorrhage into the grey matter, with or without inflammation, or of arterial obstruction. Marie is inclined to believe, that an infectious embolism or thrombosis in one of the branches of the anterior median artery, or in one of the anterior root arteries is the starting-point of the disease in acute anterior poliomyelitis of the infant. In the case mentioned on page 12 (Case F. K.), in which the disease was not limited to the anterior horns, however, the very sudden onset was suggestive of hæmorrhage or arterial obstruction. But most careful examination of the cord failed to reveal any trace of previous hæmorrhage, as above mentioned.

Goldscheider and Siemerling point out, that often in a transverse section one portion of the anterior horn is affected, whilst at a higher or lower level another part of the anterior horn is diseased. The affection is not localised to a single group of ganglion cells; one group is diseased at one level, whilst a different group is affected above or below this level. Goldscheider refers to the changes found in a case of anterior poliomyelitis, in which death did not occur for many years after the onset of the paralysis. The anterior horn was diminished in size on the affected side; the ganglion cells were almost absent and the vessel walls thickened. An examination of a series of sections showed that the degenerated ganglion cells were grouped around the altered vessels. The degeneration was not localised according to the cell groups, but according to the distribution of the vessels in the anterior horn.

Goldscheider calls attention to the changes of an inflammatory nature, proceeding from, or situated around the vessels, which occur in a number of diseases of the cord and brain. The walls of the blood vessels play an important part in the process, and Goldscheider believes that substances giving rise to cell proliferation affect the walls of the vessels by filtration and diffusion from the blood. Acute anterior poliomyelitis

is one type of this diseased process, the changes being localised to the anterior horn—to parts supplied by the branches of the anterior spinal artery. This region is the most vascular part of the cord.

Dr. Woodhead, in his interesting address at the meeting of the British Medical Association, 1894,* drew attention to the importance of the cell walls of the vessels. According to Heidenhain, the cells of the capillary walls act as true secreting cells. When stimulated there is an increased flow of lymph into the surrounding tissue, and whenever this is greatly increased there is also a wandering out of leucocytes, or, at least, an accumulation of leucocytes near the walls of the vessel. Certain substances appear to have the power of stimulating the endothelial cells of blood vessels. The product of certain micro-organisms appear to have this power.

Dr. Woodhead points out that the endothelial walls of the cerebro-spinal system of capillaries and lymphatics are extremely delicate and active; the lymph flow is great; and if the products of bacteria or bacteria themselves have any effect on the walls of capillary vessels, they should make themselves manifest here, and we should expect to find well-marked vascular and peri-vascular interstitial changes.†

The peculiarities of the lymphatics of the cord may also be of some importance in the production of the pathological changes. As above-mentioned, there are no true lymphatic vessels, but a lymph space bounded by a definite membrane ensheaths the spinal vessels.

Charcot's view that acute anterior poliomyelitis is an acute parenchymatous myelitis—*i.e.*, that the nerve cells are affected first, and the neuroglia secondarily—has had many supporters; but in a considerable number of cases in which an examination has been made at an early stage of the disease, the changes have been those of an interstitial myelitis, following the distribution of the branches of the anterior spinal artery. In other words, acute anterior myelitis, as Siemerling points out, is (in many cases at least) nothing more than an acute myelitis, with a special localisation in the area of distribution of the anterior spinal artery in the grey matter of the anterior horn. Those parts, to which the chief arteries of the cord pass, are affected, *i.e.*, the anterior horns, in which the most important groups of ganglion cells are situated. (Dauber.)

In the acute anterior poliomyelitis of infants, the changes are generally in the anterior horn of one side, either in the lumbar or cervical region; but in some cases of poliomyelitis the changes affect, at

* *Lancet*, Sept. 22, 1894.

† Woodhead. *British Medical Journal*, Sept. 22, 1894, p. 643.

one level of the cord, both anterior horns, and extend to the intermediate grey matter and to the neck of the posterior horn, *i.e.*, to the whole area supplied by the anterior median arteries, as in the case mentioned on page 12. (These cases may be described as poliomyelitis, but not *anterior* poliomyelitis, since the changes affect other parts of the grey matter besides the anterior horns. See Fig. 4).

Again,* in other cases the anterior horns of grey matter present myelitic changes on both sides, in *all* the regions of the cord; but generally they are most marked in the cervical and lumbar regions. In these cases, then, the changes affect those parts (the anterior horns) which are supplied by the chief blood vessels of the cord, the branches of the anterior spinal, through a long vertical extent (cervical, dorsal, and lumbar regions).

In a case of acute anterior poliomyelitis of the infant, reported by Redlich,† in addition to the inflammatory changes in the anterior horns, there were small inflammatory patches in the medulla, and more marked changes in the pons and crura cerebri.

(E) *In acute anterior poliomyelitis of the adult* the pathology is probably the same as that of the disease in infants, but all four limbs are much more frequently paralysed in the adult than in the infant. A spinal cord which I examined a few years ago‡ presented the early changes of the disease, death having occurred only five weeks after the onset of the symptoms. In the anterior horns on both sides, from the cervical to the lumbar region, changes were found (though they were slight in the dorsal region). There was great dilatation of the blood vessels, their sheaths were distended with round cells, and there was well marked cell infiltration (leucocytes and compound granular cells) around the vessels in the lumbar and cervical regions. The exact extent of the changes in the anterior horns varied slightly at different levels, but the chief seat of the lesion was the outer part of the anterior horns, *i.e.*, the region (K in Marie's diagram, Fig. 7) in which the anterior root arteries, and the anterior lateral artery terminate.

The changes were those of an interstitial inflammation, and followed the course of the blood vessels; they were found in the area of distribution of the *anterior spinal* system of arteries throughout the whole length of the cord.

In many sections of the *dorsal* region of the cord the vascular changes were marked, whilst other changes were slight. The vessels

* As in a case reported by Trevelyan, *British Medical Journal*, September 22, 1894.

† Redlich. *Centralblatt für innere Medicin*, No. 38, p. 896. 1894, and *Wiener klin. Wochenschrift*, 1894, No. 16.

‡ Medical Chronicle, September, 1890, p. 456.

of the anterior horns were dilated, and at the outer half of each anterior horn the perivascular sheaths were distended with round cells. The nerve cells of the outer half of each anterior horn only presented the slightest changes, and there were only a few scattered round cells just around the vessels.

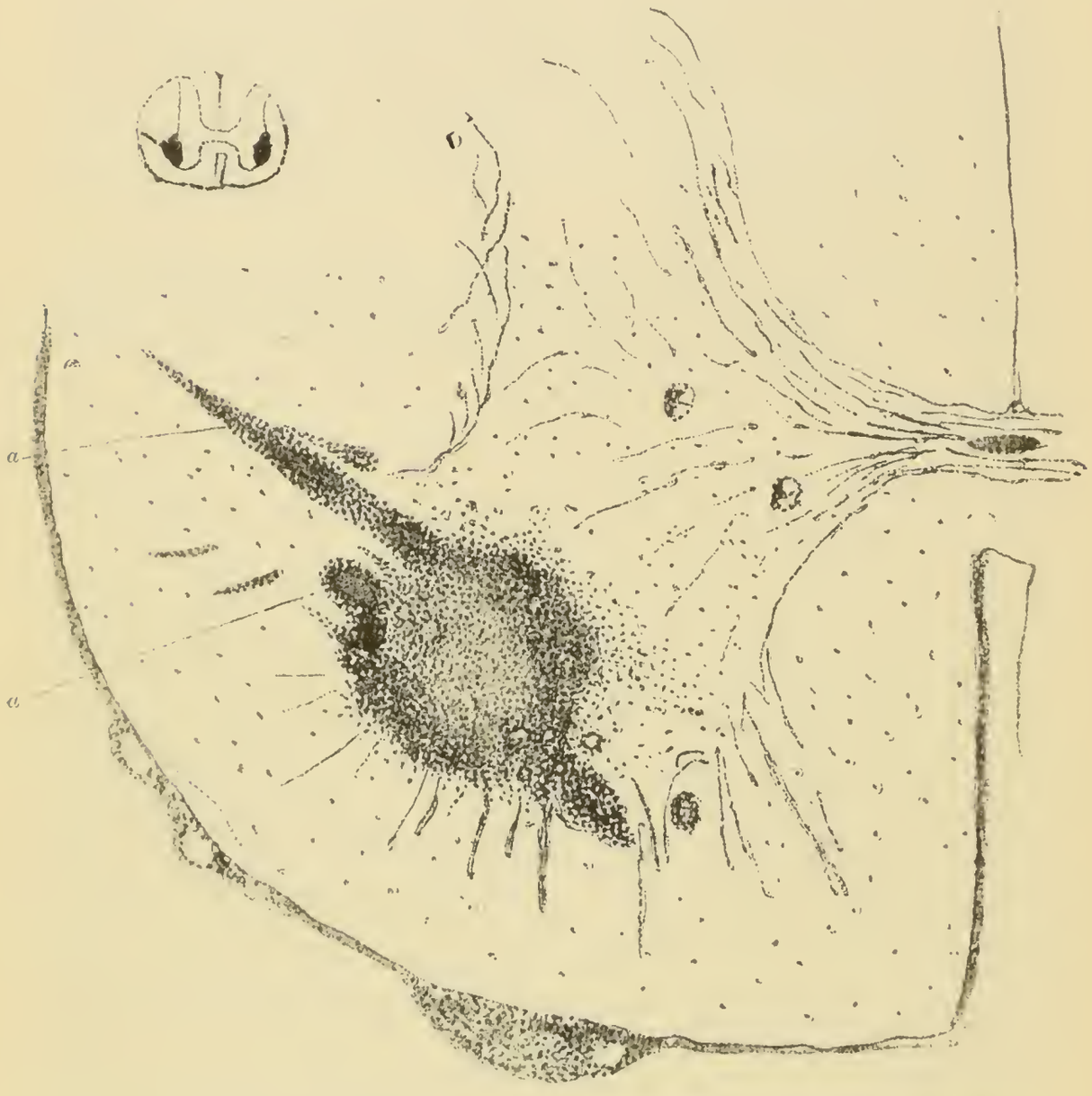

Fig. 8.—Section of Spinal Cord in a case of acute Anterior Poliomyelitis of the adult. *a a*=dilated vessels surrounded by round cells. (Logwood stain.)

In all regions the vascular changes were in great excess of the other myelitic changes. In many sections, the vessels of the anterior and posterior commissures and of the inner part of the anterior horns were also greatly dilated, and their sheaths distended with round cells, whilst the cell infiltration was limited to the outer part of the anterior horns.

The intensity and wide distribution of the vascular changes were strongly suggestive of a vascular origin of the disease, and the remarks made above with reference to the irritation of endothelial cells of small vessels would apply to the case of acute poliomyelitis in the adult.

(F) *Sub-acute and chronic poliomyelites* have been generally regarded as diseases in which the pathological changes commenced first in the ganglion cells of the anterior horn; but in a case described by Dr. Dreschfeld,* in addition to the atrophy of ganglion cells there were marked vascular changes. The blood vessels of the grey matter were dilated and filled with red corpuscles. Their walls were thickened, the middle coats in many places presenting the appearance of a clear hyaline band. Here and there in the anterior horns and grey commissures small hæmorrhages were found. From the fact that these vascular changes were well marked in the highest cervical region and also in the medulla, where as yet the changes in the ganglion cells were much less marked than in the lower cervical, dorsal, and lumbar regions, Dr. Dreschfeld is inclined to believe that the process was of a chronic inflammatory nature, with changes in the blood vessels and hæmorrhages in their neighbourhood, and that the atrophy of the motor ganglion cells was secondary to the vascular lesions. As Dr. Dreschfeld remarks, the grey matter being softer in structure and richer in blood supply, is even more likely to be affected by primary vascular changes.

Dutil and Charcot† report a case of chronic anterior poliomyelitis in which there was marked atrophy of ganglion cells in the lower cervical region of the cord. The intra-medullary arteries presented a thickening of their walls, the lumen being diminished and sometimes almost obliterated. These vascular changes appeared most constant and most advanced in the anterior region of the cord—*i.e.*, in the branches of the anterior spinal and anterior root arteries. Dutil and Charcot raise the question whether these vascular changes simply accompany the chronic changes in the anterior horns, or whether they form the primary lesion which gives rise to the sclerosis of the anterior grey columns and to the degeneration of the ganglion cells.

(G) *Disseminated Myelitis.*—The changes in the spinal cord in this disease are closely related to the distribution of the blood vessels. A typical case was recently published by Dr. Dreschfeld,‡ the pathological specimens of which I have had the opportunity of carefully examining.

* "*Brain*," Vol. VIII., 1885, p. 174—184. Dr. Dreschfeld refers to a case published by Cornil and Lépine (*Gaz. Méd. de Paris*, 1875, No. 11), which presented similar vascular changes and hæmorrhages.

† *Le Progrès Médical*, March 17, 1894.

‡ *British Medical Journal*, June 2, 1894.

The patches of myelitis in Dr. Dreschfeld's case were scattered in the most irregular manner throughout the spinal cord. During life there was double optic neuritis in addition to the spinal symptoms, and microscopically there were changes in the optic nerves. The wide and irregular distribution of the patches was in itself highly suggestive of a vascular origin. Though the patches of myelitis varied greatly in size and shape, yet in all parts the changes followed closely, and were most marked just around, the blood vessels. Where the changes were slight, they were limited to the course of the blood vessels; the latter were markedly dilated, their perivascular sheaths were distended with round cells, and in addition to these changes, there was a narrow zone of cell infiltration just around the vessels. In every section of the cord examined, even if there were no patches of myelitis, at some part of the section the vessels were dilated and their peri-vascular sheath distended with round cells. In other sections, where the changes were more extensive, there was, of course, a large area of cell infiltration around the vessels. Distinct changes were found in the optic nerve, and here, as in the cord, they followed closely the course of the blood vessels, which were greatly dilated, their lymph sheaths were distended with round cells, and just surrounding the vessels in many parts was a zone of cell infiltration.

Many cases of disseminated myelitis follow some infectious disease, others appear to be due to syphilis. The microscopical changes are very suggestive of the presence of some toxic substance in the blood (possibly bacteria or bacterial products), which acts as an irritant to the endothelium of the blood vessels, and causes an increased flow of lymph and leucocytes into the perivascular tissues, and initiates the myelitic changes.*

(H) *Acute Transverse Myelitis.*—In this disease a transverse lesion is found affecting a small vertical extent of the cord, but, in many cases, there are also slight, widely-distributed changes above and below the chief lesion (in addition to the ascending and descending secondary degeneration). These slight and disseminated changes consist of dilatation of the vessels, marked distension of the perivascular sheaths with round cells, and slight perivascular cell infiltration. Here, again, as in disseminated myelitis, the widely distributed vascular changes are very suggestive of the action of some irritant on the vessel walls.

But there is another side to the question, as to the nature of so-called transverse myelitis in certain cases. Does this localised softening or inflammation of the cord always begin as such; or is the localised softening

* See remarks on page 20.

of the cord, in some cases, the result, in the first place, of vascular obstruction, as in cerebral softening? Experiments on animals have shown that myelitis can be produced by arresting the blood supply in the cord for a short time, though the changes do not appear to correspond exactly to those of myelitis in man.

Dr. Bastian* has long maintained that in many cases of spinal softening, which are described as transverse myelitis, the changes are simply degenerative and due to disturbances of the blood supply. Can it be, as Dr. Bastian remarks, that softening in the majority of cerebral cases is non-inflammatory, whilst in the majority of apparently similar cases of softening in the spinal cord, the process is really inflammatory? As will be pointed out in a subsequent section, the distribution of the spinal vessels is such that it is improbable that embolism of the larger arteries will play any important part in the causation of spinal disease. But the vascular distribution is very favourable to the occurrence of thrombosis, and Bastian* believes that thrombosis is the cause of many cases of spinal softening which have been described as transverse myelitis. He admits, of course, that in other cases the softening is due to changes which are primarily inflammatory in origin.

Ziegler† also expresses the opinion that many cases described as myelitis are really cases of ischæmic and hæmorrhagic softening.

Nauwerk‡ has reported a case which, during life, presented symptoms of a subacute transverse myelitis in the dorsal region (complete motor and sensory paralysis of the legs with bladder and rectal symptoms). On post-mortem examination softening of the cord was found in the mid-dorsal region. Scraping from the cut surface, at the seat of softening, revealed, microscopically, fat granular cells and free fat drops. On examination of the hardened cord, marked degeneration and breaking up of the nerve fibres was found, along with extensive changes in the small vessels. There was hyaline thickening of the walls of the small arteries. A number of arterioles in the dorsal region were completely obliterated by homogeneous masses. The hyaline thrombi appeared to be due to the heaping up of white blood corpuscles. In other larger arterioles an increase of the nuclei of the intima was observed with proliferation of the endothelium, and complete or almost complete obliteration of the lumen of the vessels. Nauwerk regards the softening in this case as clearly the result of vascular obstruction, and points out that, as Kadyi has shown, the arteries entering the cord are terminal arteries.

* Bastian. "Paralyses, Cerebral, Bulbar, and Spinal."—London, 1886. pp. 557, 583.

† Ziegler. "Lehrbuch der speciellen pathologischen Anatomie." 5th Aufl. Jena, 1887 p. 361.

‡ Ziegler and Nauwerk. "Beiträge zur pathologischen Anatomie," Vol. II., Jena, 1888.

As Dr. Gowers* remarks, it is conceivable that thrombosis may occur in a minute vessel, and that the initial lesion may ultimately disappear in the intense inflammation it excites. In many cases of spinal paralysis of acute onset the paralytic symptoms commence so abruptly as to strongly indicate spinal hæmorrhage, and yet softening without hæmorrhage is found post mortem. The onset appears too sudden for primary inflammation, and hence is very suggestive of thrombosis.

The case of F. K., is a good example. The patient got up in the morning at the usual time feeling quite well, dressed, and combed her hair, then went down stairs and commenced to wind up the window blinds. She wound up the blinds of two windows, but on going to wind up the blind of the next window she found that both arms had suddenly become paralysed. The legs became affected about an hour later, and there was retention of urine. A more sudden onset can scarcely be conceived. As above mentioned, a localised area of myelitis was found in the grey matter of the lower cervical and upper dorsal regions, but though the whole of the affected area was cut into sections, and several hundreds of these carefully examined, not a trace of hæmorrhage could be detected. One can scarcely attribute this sudden paralysis to primary inflammation; the onset is strongly suggestive of a primary thrombus in a small vessel, which may have ultimately disappeared in the intense inflammation which it excited. The sudden onset and the localisation of the lesion in the case of myelitis mentioned on page 17 are also very suggestive of vascular origin.

A case which will be reported in a subsequent section shows clearly, that spinal thrombosis can produce sudden paralytic symptoms resembling those of transverse myelitis.

(1) *Disseminated Sclerosis.*—The causation of this disease is still very obscure, but a consideration of the following points throws some light on the pathology, and seems to indicate that the sclerosed patches are probably related to the distribution and lesions of the blood vessels of the central nervous system.

(1) The distribution of the sclerosed patches in the most irregular manner throughout the central nervous system, without any reference to nerve tracts or other nervous structure, whilst in adjacent parts the nervous system is normal, is a point in favour of this view.

(2) It is probable that some cases of disseminated sclerosis commence as disseminated myelitis and encephalitis.† At a later stage firm

* "Diseases of the Nervous System," p. 323, Vol. I. London, 1892.

† Ziegler. "Lehrbuch der speciellen pathologischen Anatomie."—Jena, 1887.
Leyden. *Zeitschrift f. klin. Med.*, Bd. XXI., H. 1 and 2.

fibrous tissue develops at the affected parts, and patches of sclerosis are produced. There is a pathological difference, however, between disseminated myelitis and disseminated sclerosis, though possibly it is only one of degree. In disseminated sclerosis the axis cylinders are usually present in the degenerated patches, whilst in disseminated myelitis they disappear along with other parts of the nerve fibres, and (probably for this reason) secondary degeneration (ascending and descending) occurs in the latter disease, but is generally absent in the former.

(3) At the periphery of many patches of sclerosis, more or less numerous compound granular cells are found, with indications that the morbid process is still active. Again, in some cases at the autopsy, in addition to old firm patches of sclerosis, there are soft patches containing compound granular cells both at the centre and at the periphery, *i.e.*, patches of recent development. These cases show that the morbid agent persists in the organism and is able to diffuse itself, and cause the development of new patches of the disease.

(4) Numerous observers have pointed out that in the earlier stages of the development a small artery is found in the centre of each sclerotic patch, and the chief changes are found just around the vessel.*

(5) Distinct vascular changes have been found in a large number of cases. Charcot long ago drew attention to thickening of the walls of the vessels, chiefly the external coat; when the sclerosis is marked, the vessels appear gaping and dilated on section. In many cases when the patches are of more recent development, the perivascular lymph sheaths are greatly dilated and crowded with round cells.

Ribbert† many years ago, described a case in which both old grey patches and soft greyish-red recent patches were found at the autopsy. A greatly distended blood vessel was found running through the centre of each soft patch. At two places he found a peripheral thrombosis, which consisted of white corpuscles, partially obliterating the lumen of an artery. Around the vessels in these patches, were collections of round cells. Ribbert believes, that owing to the presence of some irritating agent in the circulating blood, a clot forms at some part of a small blood vessel. This clot generally occupies only a portion of the lumen of the vessel, *i.e.*, there is only a peripheral thrombosis. At this point an irritation of the vessel wall occurs, and perivascular inflamma-

* This was first pointed out by Vulpian. With reference to vascular changes, see also—Déjérine.—*Revue de Med.*, 1884, p 193. Köppen.—*Archiv f. Psych.*, Bd. XVII., p. 63. Hess.—*Archiv f. Psych.*, Bd. XIX., p. 64. Buss.—*Deutsches Archiv f. klin. Med.*, Bd. XLV., p. 555. Popoff.—*Neurologisches Centralblatt*, 1894, No. 9.

† *Virchow's Archiv*, Bd. XC., 1882, p. 213.

tion follows. The inflammation extends around the blood vessel in a concentric manner, and invades the surrounding nervous tissues.

In a case reported by Hess,* the condition of the vessels differed at various parts. At some parts the vessels were dilated, in other parts the lumen was completely obliterated by thickening of the vessel walls. The perivascular lymph sheaths were distended within round cells and the nuclei of the vessel walls increased. Hess believes that the primary changes in disseminated sclerosis are in the vessels. As a result, white corpuscles emigrate, or if the vessels be obliterated, nutritional changes occur, the neuroglia proliferates, and the myelin of the nerve fibres disappears.

Popoff† looks upon the vessels, which are found in the centre of each patch, as the starting point of the disease. He describes the cell infiltration of the vessel walls and the emigration of the leucocytes into the surrounding tissue. He regards the pathological change as a gradual degeneration of nervous tissue around an affected vessel.

The vascular changes are most marked in the early stages of a sclerosed patch or in subacute cases. Bastian‡ states that on several occasions he has found the larger vessels of a patch of spinal sclerosis blocked by old and firm thrombi.

In a large number of cases at least, there is then distinct evidence of vascular and perivascular changes, especially in the early stages of the development of a sclerotic patch. It is interesting to note that many cases have followed some acute infectious disease, and Marie§ regards it as well established that infectious diseases (perhaps through a mixed infection) play an important part in the causation of disseminated sclerosis, and that the disease is one of vascular origin. Possibly there is some irritating agent in the circulation which stimulates the endothelium of the walls of the vessels at certain spots, and gives rise to extravasation of lymph cells and perivascular changes, or it may be that a partial or complete obstruction of small vessels occurs. Ribbert and Marie‖ look upon the changes as the result of a pathological process apparently *allied* to multiple embolism.

The vascular origin of disseminated sclerosis has, however, been disputed by several writers. In some cases the vessels have appeared normal. These have, I believe, been chiefly chronic cases or cases in which no recent patches of disease were found. If the above suggestions as to the causation of disseminated sclerosis be correct, it is quite con-

* *Loc. cit.*

† *Loc. cit.*

‡ Article on "disseminated sclerosis."—*Quain's Dictionary of Medicine.*

§ Marie. "Les Maladies de la Moelle."– Paris, 1892.

‖ Marie. *Ibid.*

ceivable, that after the acute changes have passed away the vessels may not present any marked alteration. But the evidence obtained by the examination of patches at an early condition, when the starting point of the changes would be most readily made out, is distinctly in favour of the view that disseminated sclerosis is a disease closely related to the distribution of the vessels.

A case which I examined a few years ago, illustrates very well many of the above-mentioned points.* The symptoms ran a very rapid course. A temporary numbness of one side was noticed first twelve months before death, and slight and indefinite symptoms five months before death ; but it was only after an attack of acute rheumatism that the symptoms became well defined. With the exception of the rapid course, the symptomatology was typical of disseminated sclerosis. Examination of the central nervous system revealed the usual disseminated patches of firm sclerosis, but, in addition, there were in the brain four very soft patches. The largest of these (in the right temporo-sphenoidal lobe) was of a greyish yellow colour, almost diffluent, and nearly one inch in diameter. From the cut surface a small quantity of turbid greyish fluid escaped.

Sections of the firm patches in the cord, on microscopical examination, presented the usual appearances of disseminated sclerosis. In these patches there was a marked new formation of dense neuroglia connective tissue. In the centre, the nerve fibres were absent, but at the outer part of the patches, axis cylinders were seen. The blood vessels, however, were dilated, and in many parts the peri-vascular lymph sheaths contained round cells. Though the lesions in the cord were very extensive, yet there were no tracts of secondary degeneration, ascending or descending. This is a curious point in the pathology which has often been noted.

Sections of the soft patches in the white matter of the cerebral hemispheres presented quite a different appearance. Staining according to Weigert's method showed the absence of medullated nerve fibres at these parts. The diseased patches were seen to consist almost entirely of round cells, both at the centre and at the periphery ; and there was no new formation or increase of the neuroglia connective tissue. Examination with a high power revealed the following variety of cells :—(1) Compound fat granular cells, many of which were of great size ; (2) large round or oval cells, with granular protoplasm and one nucleus—many contained fat granules ; (3) smaller round cells—leucocytes ; (4) a few nuclei surrounded by a small amount of protoplasm (neuroglia cells) ; (5) at some parts a few red blood corpuscles were mingled with the cell

* For full report see Medical Chronicle, March, 1894.

elements. The cells of the first of these varieties were most numerous. Scattered fine fibres of neuroglia connective tissue were also seen. The blood vessels were numerous and greatly dilated. Their walls were thickened with nuclei, and the perivascular sheath greatly distended—in some parts enormously distended—with round cells, leucocytes chiefly.

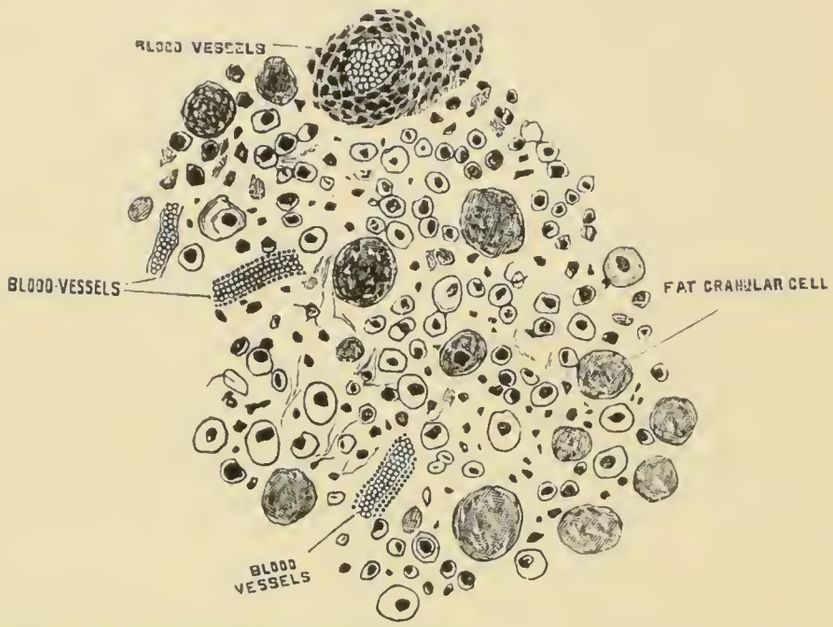

FIG. 9.—Section of the centre of a softened patch in the white matter of the cerebrum, just beneath the cortex. The section shows dilated blood vessels and cell elements. Nerve fibres are absent, and only few neuroglia fibres are seen.

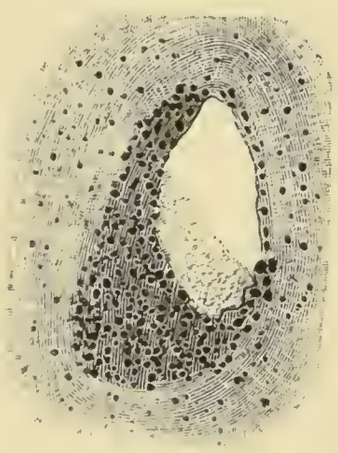

FIG. 10.—Transverse section.

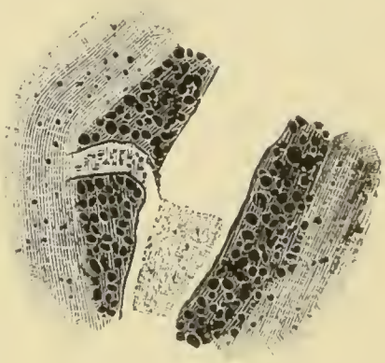

FIG. 11.—Longitudinal section.

Sections of two cerebral blood vessels, showing marked distension of the perivascular lymph sheaths with round cells.

The capillaries were greatly distended, and the nuclei of their walls increased.

The striking features in these patches were the enormous number of cells, the dilated vessels and the distention of their perivascular sheath with round cells, and the absence of any increase in the amount of the

neuroglia connective tissue. In fact, these softened patches closely resembled ordinary patches of acute softening in their microscopical appearances.

In some of the firmer cerebral patches, the appearances were intermediate between those thus described and those of the older patches in the spinal cord. They presented a slight increase of neuroglia at the

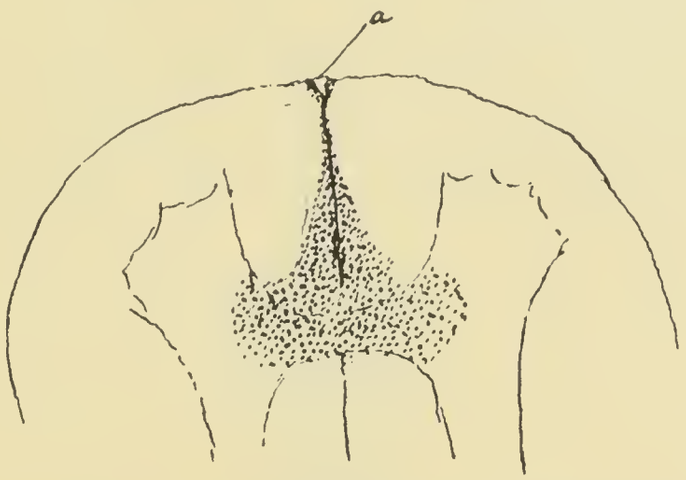

Fig. 12.—Disseminated sclerosis. Recent patch in the upper cervical region of the spinal cord in the area of distribution of the anterior median vessels. a = Thrombosed anterior median. Low power. Logwood stain).

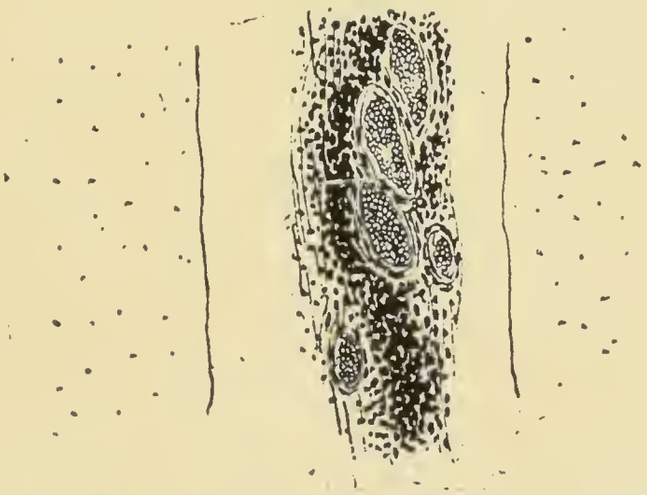

Fig. 13.—Blood vessels at the bottom of the anterior median fissure in the section represented in Fig. 12. The vessels are dilated and surrounded by numerous nuclei and round cells. (High power. Logwood stain).

centre, whilst at the outer part of the patches, cell elements were very numerous. In both varieties large compound granular cells were extremely numerous, just at the limit of the diseased area.

Some of the diseased patches in the spinal cord were densely infiltrated with round cells. In the upper cervical region there was a recent wedge-shaped patch involving the white and grey commissures,

the adjacent intermediate grey matter, and also the white matter on each side at the bottom of the anterior median fissure (see Fig. 12). This area was densely infiltrated with round cells and nuclei. It corresponded to the distribution of the vessels entering the anterior median fissure (ant. median art. and vein.): these vessels were surrounded by a dense sheath of round cells and nuclei (see Fig. 13), and were much dilated at the bottom of the fissure. Just at the commencement of the anterior median fissure (*i.e.*, at the surface of the cord), in a number of sections, a dilated vessel (anterior median vein) was seen, which contained a thrombus. In some of the sections the coagulum filled the vessel; in others there was a slight interval between it and the vessel wall (possibly this was owing to the specimen having

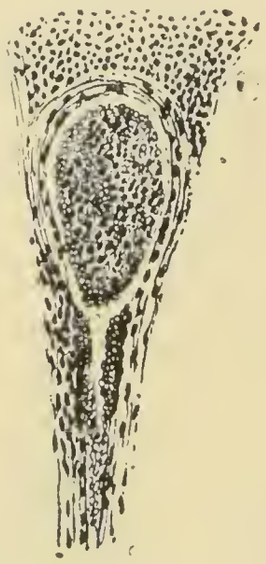

Fig. 14.—Thrombosed vessel at the commencement of anterior median fissure. (High power. Logwood stain). This is the vessel marked *a* in Fig. 12. The black dots=nuclei of leucocytes; the small circular=red corpuscles.

been in hardening fluids for three years). Sections stained with logwood showed the presence of numerous leucocytes (see Fig. 14) in the thrombus, indicating that the coagulation had occurred during life, and that the clot was undergoing organisation.

In some of the sections taken from this part of the cord, in addition to the thrombus in the anterior median vessel, there were several small thrombosed veins in the pia mater on the anterior surface of the cord.*

These thrombi are of interest in connection with the above-mentioned views of Ribbert† and Marie,‡ and the observations of Bastian§ and Ribbert.‖

* These changes have been detected since the publication of the report in the March number of the Medical Chronicle, 1894.

†*Loc. cit.* ‡*Loc. cit.* §*Loc. cit.* ‖*Loc. cit.*

Demange* reports an interesting case of sclerosis of the cord of vascular origin. During life the symptoms resembled those of amyotrophic lateral sclerosis. At the autopsy extensive atheroma of the vessels was found. The microscopical examination revealed, in all regions of the spinal cord, disseminated sclerosis. These patches were scattered most irregularly in the white matter, and in some parts penetrated the grey substance. The margins of the patches were not sharply defined, but were furnished with radiating processes (sclérose "non en plaques mais en taches.") In the centre of each small area of sclerosis was an altered artery, from which the sclerosis extended into the meshes of the neuroglia. The neuroglia was thickened and possessed numerous spider cells. The arteries appeared generally increased in number, and of unusual size. The endothelium was proliferated, the walls thickened, infiltrated with nuclei, and the lymph spaces filled with leucocytes. Demange regards the case as one commencing in a diffuse myelitis, proceeding from the peri-arteritis of the vessels—an interstitial diffuse and perivascular myelitis. He divides scleroses of vascular origin into (1) the disseminated sclerosis in patches; (2) certain interstitial diffuse scleroses, some cases being due to syphilis, others being connected with general atheroma of the vessels.

Déjérine† states that it is more than probable, that the non-symmetrical scleroses of the spinal cord are all of vascular origin.

Demange‡ also reports two other cases with similar arterial changes. From the changes in those three cases he concludes: That the vessels of the cord may be affected by a peri-arterial sclerosis, connected with general atheroma. This lesion gives rise to spots of diffuse sclerosis of vascular origin and also to miliary hæmorrhages. Clinically the symptoms are those of sclerosis of the lateral columns of the cord.

(J) *Spinal Hæmorrhage.*—Primary hæmorrhage into the spinal cord is exceedingly rare. Many cases diagnosed during life as spinal hæmorrhage have been found on post-mortem examination to be cases of myelitis or other lesion, and many cases which appear to be examples of primary hæmorrhage are cases of hæmorrhage secondary to myelitis. At first sight it appears strange that hæmorrhage should be so common in the brain and so rare in the cord, but a consideration of the spinal vascular distribution will do much to explain this difference. As already pointed out, the spinal cord is supplied by long narrow

* "Contribution a l'étude des scléroses médullaires d'origin vasculaire."—*Revue de Médecine*, 1884, p. 753.

† "Etude sur la sclérose en plaques cérébro-spinale à forme de sclerose laterale amyotrophique."—*Revue de Médecine*, 1884, p. 193.

‡ *Revue de Médecine*, 1885, p. 1.

and tortuous arteries, and hence the vessels are not subject to the high pressure, which is such an important cause of degeneration and rupture of the arteries of the brain; also miliary aneurisms, the rupture of which is such a frequent cause of cerebral hæmorrhage, are almost unknown in the spinal cord.* Further, there is more supporting connective tissue around the blood vessels in the spinal cord than in the brain.

When hæmorrhage does occur in the spinal cord, except in traumatic cases, the blood extravasation begins in the grey matter and generally remains limited to it. The hæmorrhage may extend for some distance upwards and downwards in the grey matter; only when it is very great does the white matter become involved. This localisation we can readily understand, because the grey matter is the softest part of the cord, also it is more vascular than the white, and the vessels of the grey matter have less external support than those of the white.

(K) *Spinal Embolism.*—From the vascular distribution it is not to be expected that embolism would be of much importance in the causation of spinal disease. Anastomosis of the spinal arteries on the surface of the cord is so free, that it is improbable that embolism would give rise to any great area of softening. Again, the spinal arteries are all of small size. As Bastian points out, there are no large arteries like the middle cerebral, coming off more or less directly from one of the great vessels of the arch of the aorta, and supplying a large area of nerve tissue. Emboli are conveyed to the brain much more rarely by the vertebrals than by the carotids, and the largest arteries of the cord (anterior and posterior spinal) are derived from the vertebrals. Apart from these arteries, the smaller twigs from the intercostals and lumbar arteries are still less likely to convey embolism. Hence, it is not surprising that the evidence of spinal embolism, as a cause of spinal disease, is so slight. It must be remembered, however, that the small arteries entering the cord are terminal arteries, according to Kadyi, and a few cases have been recorded of patients suffering from valvular disease of the heart, who have been suddenly attacked with paralysis in the legs. Probably some of these have been cases of spinal embolism.

It was shown experimentally by Flourens, Panum, and Vulpian long ago that by the injection of inert powders embolism of the arteries of the cord could be produced, and loss of the functions of the cord was the result. In two papers recently published (whilst this reprint was passing through the press) Lamy† has shown that the foci of hæmorrhagic softening, following the obstruction of small spinal arteries by the injection of inert powders, always occur first in the grey substance.

* One case is recorded, however, by Leyden.

† *Archives de Neurologie*, December, 1894.
Archives de Physiologie normale et pathologique, No. 1, 1895.

(L) *Spinal Thrombosis.*—Whilst the vascular distribution in the spinal cord, as above mentioned, is so unfavourable to the occurrence of hæmorrhage and embolism, it appears to be distinctly favourable to thrombosis. The small narrow arteries, their long and tortuous course, especially in the lower part of the cord, the free anastomosis on the surface of the cord, the consequent low blood pressure and probable slowness of the blood current, all predispose to thrombosis. Then again the tortuous network of veins, which surrounds the spinal cord and dura mater, will probably give rise to slowness of the blood current and so predispose to thrombosis.

As Dr. Bastian* points out, it is somewhat remarkable that spinal softening is generally looked upon as inflammatory in origin, whilst cerebral softening, a condition apparently similar, is known to be due to vascular obstruction in most cases. It is very improbable that spinal softening should be always inflammatory, whilst cerebral softening is nearly always due to vascular obstruction. These considerations would lead one to expect, that thrombosis would play an important part in the causation of spinal diseases, but as yet the pathological evidence has been very small. As above mentioned, Dr. Bastian believes that many cases of softening, described as cases of myelitis, are really due to thrombosis.

The microscopical examination of a spinal cord, which I made a short time ago, showed clearly that paralysis and other symptoms of a transverse lesion of the cord can be produced by *spinal thrombosis.* The specimen was one which was exceedingly favourable for examination of the vessels, as the changes were comparatively recent, death having occurred only fifteen days after the onset of the paralysis.† The patient, T. L., was under the care of Dr. Steell at the Manchester Royal Infirmary. The history was, that for one month he had suffered from slight pain in the back. Then retention of urine occurred suddenly. In the evening of the same day his gait became somewhat unsteady. Next morning, on awaking, he found that both legs were completely paralysed. On examination at the Infirmary, two days later, the legs were completely paralysed, there was complete anæsthesia (to tactile and painful impressions) up to the level of the sixth intercostal space, with bladder and rectal symptoms. There was a distinct history of syphilis two years previously. In spite of treatment with mercury and iodide of potash, he rapidly became worse; cystitis and bed-sores developed, and death occurred 15 days after the onset of the paralysis. After hardening the cord in Müller's fluid, marked naked eye changes

* "Paralysis, Cerebral, Bulbar, and Spinal," p. 551. London, 1886. "Quain's Dictionary of Medicine" Vol. II. Section Spinal Cord. London, 1885.

† For details of the case, see papers by the writer in the *Lancet*, July 7th, 1894.

were seen on section for about $4\frac{3}{4}$ inches in the middle of the dorsal region. Microscopical examination revealed extensive syphilitic disease of the arteries and veins of the cord and meninges—endarteritis and peri-arteritis, endophlebitis, and periphlebitis. These changes were best seen in the vessels of the pia mater, and were most marked in the mid-dorsal region, though they were also present in the cervical and lumbar regions. There was also leptomeningitis, best seen in the dorsal region. At the part where the naked eye changes could be detected (middle dorsal region), microscopical examination showed that the transverse sections were studded with great numbers of enormously dilated blood vessels. Many of these exhibited marked thrombosis, especially in the grey matter and in the adjacent part of the lateral columns of white matter. The walls of the vessels were thickened and their nuclei were greatly increased in number; the tissues around for a considerable distance were infiltrated with round cells and the number of nuclei greatly increased. In many places there was considerable hæmorrhagic infiltration in the grey matter around the thrombosed vessels. At one point almost the whole of the grey matter of one side was infiltrated with blood.

Fig. 15 represents the appearance of a section of the cord at the mid-dorsal region.

Figs. 16, 17, and 18 represent the changes in the arteries and veins.

Figs. 19 and 20 represent thrombosed spinal vessels.

At some parts the vessels of the meninges were thrombosed also.

The main changes found at the most diseased part of the cord were, therefore (1) thrombosis and great dilatation of the vessels; (2) inflammatory changes—increase of nuclei and cell infiltration in the walls of the vessels, and in a wide zone of the surrounding tissues; (3) hæmorrhagic infiltration around the vessels where the disease was most intense.

Microscopical examination showed clearly that the primary change was syphilitic disease of the blood vessels, and that this had been followed by thrombosis and hæmorrhagic infiltration. There can be no doubt that the thrombosis of blood vessels occurred before the hæmorrhage, and not as a result of it, because—(1) though the thrombosis was well marked in the middle dorsal region at the seat of hæmorrhage, yet thrombosed vessels were found for some distance above and below, and also in the meninges far away from the hæmorrhage (in the cervical and lumbar regions); (2) in most of the sections in the middle dorsal region the chief features were the thrombosed and dilated vessels, whilst the surrounding hæmorrhagic infiltration was comparatively small, and in some sections absent; (3) many of the thrombi were of older date than the hæmorrhage; (4) the blood corpuscles in many of the thrombosed vessels were broken down into granules, and the clot partially organised,

Fig. 15

Fig. 15.—Transverse section of spinal cord (dorsal region) in the case (T. L.) of acute syphilitic paraplegia. (Low power. Diagrammatic. Stained with logwood and eosin). The diagram shows the numerous enormously dilated vessels, most of which are *thrombosed*. The surrounding cell infiltration and increase of nuclei are represented by dots. The parts shaded with parallel lines are infiltrated with red blood corpuscles.

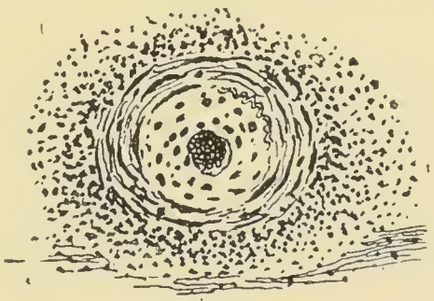

Fig 16

Fig. 16.—Meningeal artery, showing well-marked syphilitic endarteritis and peri-arteritis. Case (T. L.) of acute syphilitic paraplegia. (High power. Logwood and eosin.)

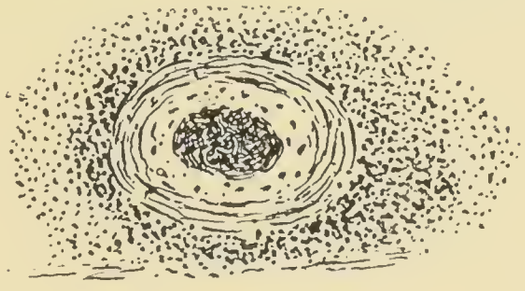

Fig 17

Fig. 17.—Meningeal vein from the same case, showing endo- and peri-phlebitis. The lumen is obstructed by a thrombus. (High power. Logwood and eosin.)

whilst the corpuscles in the hæmorrhage around were unaltered, and evidently the hæmorrhage was more recent than the thrombus; (5) there were marked syphilitic vascular changes which alone would account for the thrombosis.

(M) *Spinal Syphilis.*—Syphilis, as is well known, gives rise to important changes in the blood vessels. The cerebral arteries are especially liable to be affected, but changes may occur in the small vessels of almost any organ. In this syphilitic arteritis the arterial coats which suffer most are the adventitia and the intima (syphilitic endo-arteritis).

In the brain, syphilitic disease of the arteries is very frequently followed by thrombosis, and the consequent extensive cerebral softening and marked cerebral symptoms. In the spinal cord, there are, of course, no large arteries, the obstruction of which would be followed by an extensive area of softening, as in the case of the middle cerebral arteries and their branches. But the arteries *within* the cord are terminal arteries, and it is only reasonable to expect, that if the spinal arteries were affected by a syphilitic arteritis, of the same nature as that of the cerebral arteries, spinal softening or degeneration would be produced, and that paralytic symptoms would follow. The case T. L., just described on page 35, is a clear example of paraplegia due to syphilitic disease of the spinal vessels.

Clinically there are several types of syphilis of the spinal cord :—

(1) The symptoms may be those of a tumour and pathologically due to a syphilitic *gumma.*

(2) Syphilis may give rise to spinal meningitis. Or the symptoms may commence with pain in the back and indications of meningeal affection, which are followed at a later period by incomplete paralytic symptoms, indicative of implication of the cord. The pathological condition is one of meningo-myelitis. This is a common form of spinal syphilis—according to some authors the most common form. The arteries and veins of the meninges often show marked syphilitic changes (periarteritis and periphlebitis). According to Lamy* the meningeal lesions sometimes appear less important than the accompanying lesions of the vessels. He points out that the veins are affected first, and generally, even at a late stage, they are more affected than the arteries. Also in the cases most favourable for examination, the commencement of the lesion is seen to be the tunica adventitia of the vessels.

(3) In another group of cases the symptoms are those of acute spinal paralysis (acute transverse myelitis).

* Lamy (H.). "De la méningo-myélite syphilitique."—*Nouvelle Iconographie de la Salpêtrière,* Nos. 2, 3, 4 and 5, 1893.

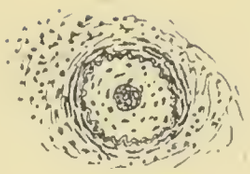

Fig 18

FIG. 18.—Small spinal artery, showing marked syphilitic endarteritis and peri-arteritis from the case (T. L.) of acute syphilitic paraplegia. (High power. Logwood and eosin.)

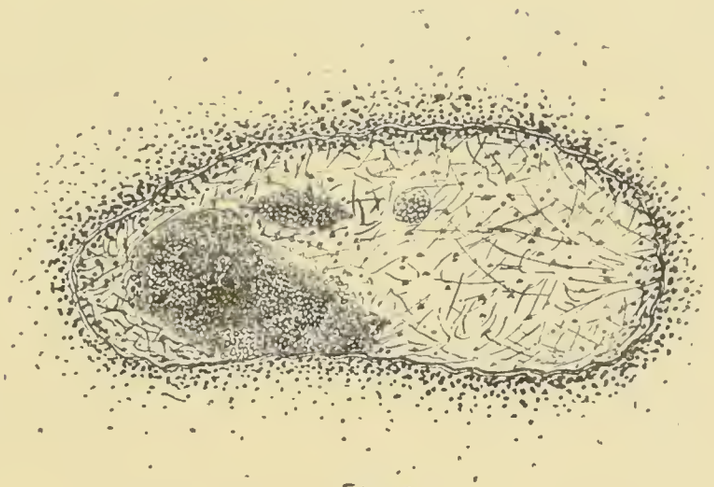

Fig 19

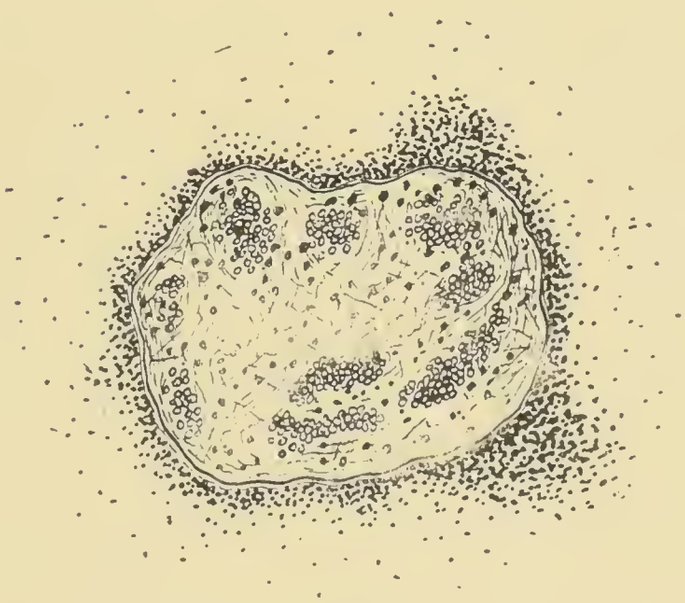

Fig 20

FIGS. 19 and 20.—Thrombosed spinal vessel, case (T. L.) of acute syphilitic paraplegia. These are small vessels from the grey matter and adjacent white matter of the cord in the dorsal region. They have become enormously distended, and the greater portion of the lumen of each is occupied by the organised part of the thrombus. The small circles=the remaining red blood corpuscles. The black dots=nuclei. (Logwood and eosin.)

In many of these cases, the pathological changes appear to have been the same as those of acute transverse myelitis from other causes. Possibly in some of these cases, myelitis has not been produced by syphilis, but has simply occurred in persons who have previously suffered from syphilis. But in a number of cases examined within recent years, very marked syphilitic *vascular* changes have been found.

The pathological changes in the case T. L. (mentioned under Thrombosis, Section L) show clearly that in syphilitic patients *acute spinal paralysis*—symptoms of a *transverse lesion of the cord* of acute onset—can be produced by *syphilitic disease of the vessels, followed by extensive thrombosis*, with perivascular inflammation and hæmorrhagic infiltration.

Schmaus* has reported a case of paraplegia of somewhat sudden onset, in a syphilitic patient, in which post mortem revealed :—(1) Irregularly scattered patches of sclerosis in the white substance, degeneration in Goll's column in the cervical region, and sclerosis of the peripheral part of the cord ; (2) Hyaline fibrous thickening of the intima of the arteries, with inflammatory infiltration of the whole vessel walls ; in the veins similar changes—phlebitis and obliterating endophlebitis. (3) Proliferation of the neuroglia in the neighbourhood of the vessels. The spinal disease followed the course of the vessels. In many of the small vessels thrombi were found.

Sottas† has reported the changes in the cord in three cases, in all of which extensive syphilitic vascular changes were found with narrowing or obliteration of the arteries. Sottas concludes, that in syphilitic paraplegia, the primary alteration is a vascular lesion which produces ischæmia and softening.

Déjérine‡ concludes from the result of the pathological examination of four cases, that the so-called acute transverse myelitis of syphilitic patients is really a softening of the cord produced by endarteritis—just as the hemiplegia of syphilitics is so often produced by cerebral softening due to arteritis. Whilst admitting that the lesions of spinal syphilis may be produced in other ways, the pathological examination in his four cases showed clearly that the ordinary forms of syphilitic paraplegia of acute onset were the result of spinal softening due to endarteritis. The softening may be followed by sclerosis at a later date.

Knapp§ has reported a case of acute ascending motor and sensory paralysis with ocular and bulbar paralysis in a syphilitic patient. Pathological examination revealed softening in the dorsal and lumbar regions.

* *Deutsches archiv. f. klin. Med.*, Bd. XLIV., Heft 2 and 3, 1889.

† *Compt. Rend. Soc. de Biolog.*, 1893, April 15.

‡ *Comp. Rend. Soc. de Biolog.*, 1893, p. 432.

§ Quoted by Rumpf. "Syphilitische Erkrankungen des Nervensystems."—Wiesbaden, 1887, p. 349. Also *Neurolog. Centralblatt.*, 1885, No. 21.

The arteries showed marked hyaline thickening of the intima, with narrowing of the lumen. The veins were distended, full of blood, and surrounded by numerous round cells.

(4) In other cases the symptoms of spinal syphilis are those of a *chronic myelitis.*

Greiff* has published a case of paraplegia of somewhat gradual onset in a syphilitic patient. Pathological examination revealed extensive inflammation of the pia mater. The arteries and veins presented marked changes—endo-arteritis and phlebitis obliterans. There was swelling and hyperplasia of the neuroglia interstitial tissue, with inflammatory exudation around the vessels, and moderate implication of the nervous elements.

Rumpf† has reported a case of cerebro-spinal syphilis, in which the spinal symptoms were due apparently to syphilitic disease of the vessels of the cord.

Erb‡ has called attention to a form of spinal paralysis, very frequently met with in syphilitic patients. The onset of the symptoms is gradual. The disease commences with loss of power in the legs, which passes on to marked spastic paresis, but seldom to complete paralysis. The knee-jerks are increased, but there is only relatively slight muscular rigidity or contracture. There are bladder symptoms, and sensation is affected, but, as a rule, only slightly. Under treatment, improvement occurs in at least half the cases.

Erb thinks the disease is due to a partially transverse lesion of the cord—*i.e.*, one not affecting the whole transverse section of the cord. The posterior halves of the lateral columns, the posterior grey matter, and the posterior white columns he believes to be chiefly affected.§ As regards the nature of the changes, Erb believes that they consist partly in a syphilitic infiltration of the cord, partly in myelitic changes (degenerative) which have followed syphilitic arterial disease.

Some time ago|| I had the opportunity of examining the spinal cord in an old case of syphilitic paraplegia (of 9 years' standing). The onset had commenced somewhat suddenly with retention of urine followed by paraplegia. Rapid recovery occurred, but a few weeks later the paraplegia returned, and this with bladder and rectal symptoms continued up to the patient's death, 9 years afterwards. Marked rigidity

* Grieff. *Archiv. f. Psychiatrie*, Bd. XII., p. 564.

† Rumpf. "Syphilitische Erkrankungen des Nervensystems."—Wiesbaden, 1887, p. 341.

‡ Erb. *Neurologisches Centralblatt*, No. 6, 1892. A number of papers have recently appeared on this subject: Kuh. *Deutsche Zeitschrift für Nervenheilkunde;* Turner. *The Lancet*, May 5, 1894; Clarke. *The Lancet*, May 26, 1894.

§ Oppenheim (*Berliner klin. Woch.*, No. 35, 1893) believes that Erb's syphilitic paralysis is due to ordinary syphilitic meningo-myelitis in the dorsal region, and that it is not a disease *sui generis*; but he admits the frequency of the group of symptoms which Erb has described.

|| See Medical Chronicle, July, 1891.

of the legs with contractures gradually developed. On examination of the cord, sclerosis of the lateral columns was found (in all regions), with sclerosis of Goll's column in the cervical region and sclerosis of the periphery of cord in the lower cervical region. There was old meningitis in the cervical region. At no part was there any indication of an old, completely transverse, myelitis. The striking feature of the diseased parts was the enormous thickening of the walls of the blood vessels, which was very suggestive of the vascular origin of the disease. The adventitial coat was chiefly affected. Of course, in all forms of sclerosis the vessels are often thickened, but I have never seen thickening of the vessel walls so marked in other forms of sclerosis of long standing.

(N) *Locomotor Ataxia.*—Buzzard,* Adamkiewicz,† Sachs,‡ and others§ have drawn attention to the arterial changes in the cord in some cases of locomotor ataxia, and it has been suggested that vascular disease is, in certain cases, the cause of the degenerative changes in the nervous elements. A number of cases have been recorded which have presented symptoms of locomotor ataxia during life, whilst on pathological examination syphilitic infiltration, interstitial myelitis, syphilitic changes in the vessels and meninges, etc., have been found in the posterior part of the cord.‖ But in the majority of cases of tabes the evidence with regard to the dependence of the cord lesions on vascular disease is not conclusive.

The observations of Leyden, Marie, Dejérine, and others, tend to show that the changes in the cord in tabes are secondary to disease of the posterior nerve roots. This disease of the posterior roots has been attributed by Marie to an altered condition of the cells of the posterior ganglia, but only very slight microscopical changes were detected by Wollenberg in fourteen cases. According to Obersteiner and Redlich,¶ however, the disease of the posterior roots commences just at the point where they pass through the pia mater to enter the cord, and is the result of a chronic meningitis and arterial sclerosis at this region.

It is a point of some interest to note, that in locomotor ataxia the changes in the cord, and in the posterior nerve roots also, occur first, and are most marked, in the lumbar region in the majority of cases. Now this is the region which, according to Dr. Moxon (as above

* *Brain*, Jan., 1884.

† Adamkiewicz. *Arch. f. Psych.*, Bd. X., Heft 3.

‡ Sachs. *New York Med. Jour.*, Jan. 6, 1894.

§ Krauss. *Arch. f. Psych.*, Bd. XXIII., p. 389.

‖ Dinkler. *Deutsche Zeitschrift f. Nervenheilkunde*, 1893, III., 4 u. 5. Abstract. *Neurologisches Centralblatt*, June 15, 1893. Ewald. *Berliner klin. Wochenschrift*, No. 12, 1893.

¶ Obersteiner and Redlich. *Neurologisches Centralblatt*, June 15, 1894 (Abstract).

mentioned) is most feebly supplied with blood. Further, the blood supply to the posterior part of the cord and posterior roots is probably feebler than that of the anterior parts.

(O) *Paralysis Agitans.*—A number of recent records, of the careful pathological examinations of the nervous systems in paralysis agitans, have shown, that fairly well defined changes are found in the spinal cord. In the papers referred to below* we have the records of fourteen recently published cases, and in these, well marked vascular and perivascular changes were found in the spinal cord :—chiefly thickening of the walls of the blood vessel, perivascular sclerosis or inflammation, and diffuse interstitial sclerosis, starting from the blood vessels and the pia mater. Thickening of the walls of the spinal blood vessels occurs in old people, but as Redlich points out, it never becomes so marked as in paralysis agitans, also the perivascular sclerosis is not met with in the spinal cords of senile persons. Though these changes may not explain all the symptoms of paralysis agitans, they are nevertheless sufficiently constant to show, that, as far as the spinal cord is concerned, the disease has a definite pathological anatomy, consisting of vascular and perivascular lesions.

To furnish conclusive evidence with regard to the causation of most diseases of the nervous system is a very difficult task, and I cannot hope that *all* the views brought forward will stand the test of future research. Still, I think, this review will have brought together evidence of some importance, with respect to the relations of spinal diseases to the distribution and lesion of the spinal blood vessels. The subject does not appear to have received so much attention as it deserves, yet it is of importance not only pathologically, but also with regard to the treatment of spinal diseases, since a more exact knowledge of the causation of these obscure affections ought to lead, in course of time, to more successful therapeutics.

* Koller. *Archiv f. path. Anatomie und Physiologie und f. klin. Medicin*, Virchow, Bd. CXXV., 1891.

Ketscher. *Zeitschrift für Heilkunde*, Bd. XIII., Heft 6, 1892, and *Neurologisches Centralblatt*, No. 5, 1893.

Dana. *New York Medical Journal*, June 10, 1893.

Redlich. *Jahrb. f. Psychiatrie*, Bd. XII., and *Neurologisches Centralblatt*, July 1, 1894.

[An abstract of these papers by the writer was published in the Medical Chronicle, August, 1894.]

John Heywood, Excelsior Printing and Bookbinding Works, Manchester. [P 2154